Indoor Air / Volume 32, Issue 5 / e13025

EDITORIAL | Open Access

Systematic way to understand and classify the shared-room airborne transmission risk of indoor spaces

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First published: 22 May 2022
https://doi.org/10.1111/ina.13025
Citations: 1

The COVID-19 pandemic has brought a new appreciation of the importance of airborne disease transmission. Airborne transmission is caused by the inhalation of pathogen-containing aerosols that are produced by an infected person.[1] Before the pandemic, the main accepted airborne diseases were tuberculosis, measles, and chickenpox. At the start of the pandemic, WHO concluded that COVID-19 was a contact/droplet/fomite disease, understood to mean either direct physical contact, or a spray of ballistic larger particles that impact on eyes, nostrils, or mouth, or are picked up by hands and delivered to the same body parts.[1, 2] However, it has become clear that COVID-19 really is a predominantly airborne disease.[3, 4] A re-examination of literature evidence also concluded that all or almost all transmissible respiratory diseases are airborne, including influenza, SARS, MERS, and rhinovirus.[5] In hindsight, this is not so surprising: respiratory diseases infect the respiratory system, and aerosols of pathogen-containing respiratory fluid and saliva are generated when breathing, talking, singing, shouting, coughing, or sneezing. Inhalation of those aerosols leads to their deposition in the respiratory tract of susceptible people, potentially initiating infection.[5] A smaller fraction of COVID-19 transmission may occur through either deposition of respiratory aerosols on the eyes, large droplet spray, direct contact (e.g., kissing), or through inhalation of aerosols containing fecal material or emitted from resuspension of fomites. In principle, surface touch (fomites) can also lead to infection, although with low probability.[6]

1 THE EMISSION PLUS DILUTION DOMAINS OF AIRBORNE TRANSMISSION

There are four spatial domains of airborne transmission:

a. Indoors in close proximity, where the respiratory jet of the infected is inhaled by the susceptible with limited dilution.
b. Indoors in a shared-room, where the exhaled air is substantially diluted into a room, and is inhaled over time by people who are not in close proximity to the infected person.
c. Longer-range indoors, where the infected and susceptible do not occupy the same room at the same time.
d. Outdoors.

Note that much of the literature lumps (b) and (c) under “long-range”, but this finer classification[7] is advantageous to the discussion. Transmission cases from all of these modes have been documented for COVID-19. Close proximity airborne transmission is known to be important based on contact tracing, and is reduced with measures such as physical distancing.[8, 9] Shared-room airborne transmission appears to be the dominant cause of superspreading events.[7, 10] Contact tracing is typically only performed for close proximity situations, which has led some researchers to posit that shared-room superspreading is minor. However, this “lamppost science”[11] is contradicted by extensive evidence that show that superspreading has played a major role in the spread of the pandemic.[7, 10] Longer-range transmission appears to be less common,[9] but it has been documented multiple times in, for example, quarantine hotels and apartment buildings.[12] Outdoor transmission is possible but much lower than for indoor locations[9, 13] despite similar or smaller interpersonal distances,[14, 15] and typically only observed in close proximity.

The same patterns are observed for other airborne diseases, with those less contagious (e.g., tuberculosis) favoring transmission in close proximity. Less transmissible airborne diseases are less prone to shared-room superspreading or longer range transmission, but these can be observed especially for long exposure times in poorly ventilated environments.[16] In contrast, the most transmissible diseases (e.g., measles and chickenpox) cause more frequent shared-room superspreading and longer range events.[17]

This pattern for different airborne diseases and distances/dilutions can be generally understood by the dominant impact of just three factors, whose understanding guides the avenues of intervention:

i. Different diseases differ greatly in the average rate of emission of airborne pathogen infectious doses (“quanta”, defined as the amount of pathogen inhaled that will result in infection for 63% of susceptibles) for individuals performing a given activity. Largest emission is observed for measles, intermediate for COVID-19, and lower for tuberculosis.[7, 18] The emission level also changes substantially across different infected individuals and different activities.
ii. The likelihood of transmission is strongly modulated by the effective dilution of the

exhaled air before it is inhaled. Increasing dilution characterizes the transition from close proximity (lower dilution) to shared-room air (medium) to longer range (high in most situations) to outdoors (very high under most situations, except in close proximity).[9]

iii. Exposure time.

The quantitative analysis of the interplay of emission strength, dilution, and time requires a sufficiently accurate characterization of the disease. Many COVID-19 superspreading events have been documented in the literature with all of them being potentially explained by airborne transmission. To the best of our knowledge, no peer-reviewed analysis of any individual superspreading event has supported non-airborne transmission. Recently, Peng et al.[7] have shown (Figure 1A) that all the literature events that had complete information for airborne investigation are consistent with shared-room superspreading (with the assumption that air in the room mixes faster than the duration of the event). Their quantitative analysis, which is consistent with the emission estimates of Mikszewski et al.,[18] allows extending the estimation of the probability of infection to other conditions.

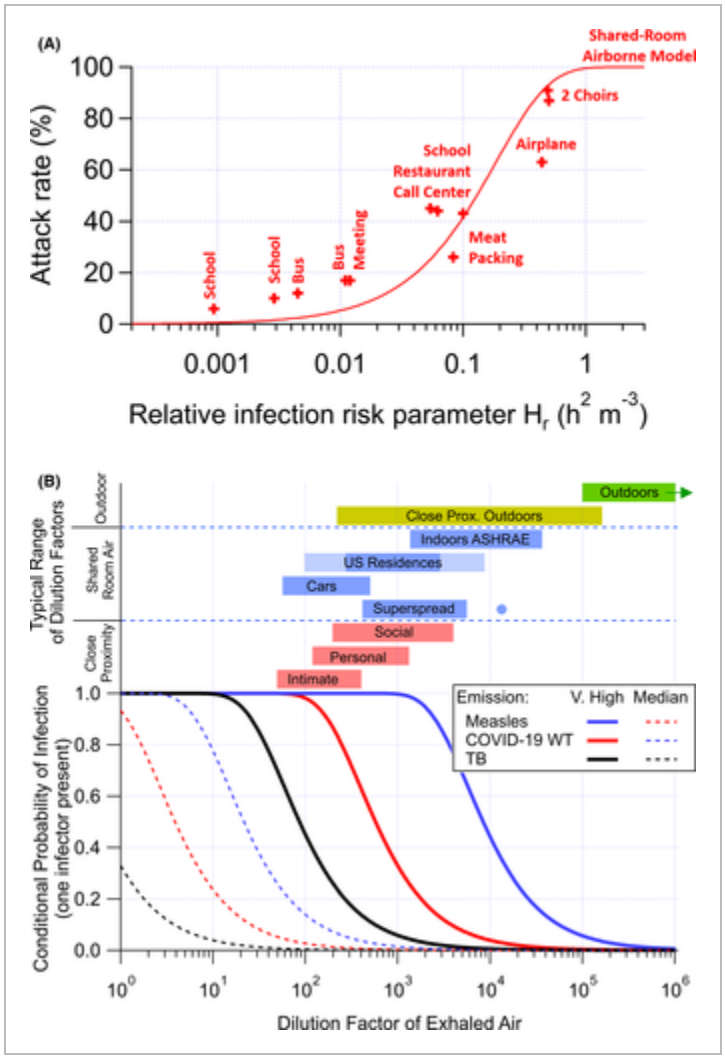

FIGURE 1

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(A) Attack rate (i.e., the fraction of susceptible people who are infected in a given event) of COVID-19 (wild type) in literature superspreading events vs. the relative infection risk parameter (see text); adapted from Peng et al.[7] Note that the model assumes that a single infector was present, which is appropriate for these superspreading events. (B) Typical ranges of dilution factors for different shared-room air, close proximity, and outdoor situations; and estimated probability of airborne transmission for each susceptible individual present for a 1 h exposure to an infected individual for different diseases (solid line: typical very infective individuals from superspreading studies in the scientific literature; dashed line: typical infected individuals with median emission rates) using the Wells–Riley infection model[7]; emission rates from Refs. [7, 18] dilution factor ranges for close proximity and shared-room air situations estimated from Refs. [7, 20, 21] and CO_2 measurements in our laboratory; COVID-19 WT refers to the wild type SARS-CoV-2 variant from the early epidemic, values are higher for omicron BA.1 variant by an estimated factor of ~2.5. See **Supplementary Information** for the choice of the values shown in the graph. Versions of this figure with a logarithmic vertical axis, with different exposure times, with an estimate of the SARS-CoV-2 omicron variant, and with some example mitigations are shown in Figures SI-3 to SI-6 in the **Supplementary Information** section

Figure 1B illustrates the interplay of pathogen emission strength and effective dilution (for a constant exposure time) on the probability of airborne infection. Figure SI-4 in the **Supplementary Information** illustrates the effect of exposure time. These figures are consistent with the following observed epidemiological trends:

- Transmission probability decreases with increasing distance between people in close proximity.[8]
- Many infected people do not transmit to anyone, but a minority (likely those with higher pathogen emission and present in lower dilution and/or higher exposure time situations) can infect many others.[10]
- For shared-room transmission, different location and activity combinations differ greatly in their inherent risk.[7] Superspreading occurs in less-ventilated indoor environments where pathogen emission rates are also high.[7, 9, 13]
- Any airborne disease for which infection in shared-room air is observed will be much more contagious in close proximity.[16, 17, 19]
- Although transmission probability is highest in close proximity, more people are present in shared-room situations than in close proximity, and thus both modes of transmission are important.[7, 10]
- The trend is consistent with the decreasing transmissibility and superspread as the pathogen emission strength decreases from measles, to COVID-19, and tuberculosis.[7]

- Transmission in close proximity and shared-room air can be reduced by ventilation and filtration, which increase the effective dilution ratio of exhaled pathogens significantly.
- Masks reduce transmission by providing additional effective dilution of the pathogen content of exhaled and inhaled air. This reduction can reach 1–2 orders of magnitude for high-filtration well-fit masks.
- Transmission at long-range is less frequent due to much higher dilution, and it may be observed mostly when “unlucky” air paths with lower dilution and longer exposure times are present,[12] for example, with stack flow in apartment buildings.
- Transmission outdoors in close proximity is less frequent than indoors due to higher dilution driven by stronger outdoor air movements. Outdoor transmission is not expected or observed beyond close proximity.[9, 13]
- The increase of transmission risk associated with vocalization and physical activity[7] is substantially driven by the increase in pathogen emission rates (and to a lesser degree, of inhalation rates) during these activities. Quiet breathing is expected to trend closer to the median cases in the graph, and loud or strong exercise activities are more likely to lead to very high emission rates. For example, many superspreading events have been identified in choirs,[7] but none to our knowledge in libraries or movie theaters where quiet sedentary breathing is the norm.

Li et al.[19] recently proposed that poor ventilation also worsens close proximity transmission, as the room air that is entrained (to dilute the just-exhaled air) is accumulating pathogen-containing aerosols over time. This effect is clear in Figure **1B**, as the room-level dilution rates in poorly ventilated situations provide a strong upper limit to the dilution ratio in close proximity, that is, dilution of exhaled air in close proximity cannot achieve a lower pathogen concentration than that present in the dilution air (room air).

However, the prominence of infection in close proximity led to the classification of all airborne diseases as “large droplet diseases” for many decades, and of COVID-19 during much of the pandemic, due to a long-held historical error.[16, 17, 22] For example measles, now considered the prototypical airborne disease by the medical community, was classified as a droplet disease until the 1980s, due to ease of infection in close proximity, and documented cases of lack of infection in shared-room air.[17] We see from Figure **1b** that those epidemiological patterns are entirely consistent with close proximity airborne infection, combined with less frequent shared-room superspreading observed in *some* but not all cases (only when a higher emitting individual is present in locations with less ventilation), and longer range transmission being much less common, and only likely to be observed for a combination of high emitters, lower dilutions, and longer times.

Thus ease of infection in close proximity, less infection with increasing distance, and superspreading with sensitivity to ventilation or filtration and exposure time should be

interpreted as a *likely signature of airborne transmission*, and not as “droplet transmission” as traditionally done. “Droplet transmission” *misinterpreted the effect of dilution as the effect of gravity*, which would reduce transmission by making sprayed droplets fall to the ground near the infected person. The distinction is critical to understand the epidemiological patterns of transmission and the mitigations. Some in the medical community have mistakenly only considered “airborne” those diseases for which infection at longer range could be documented, but such a characterization is inconsistent with both physics and epidemiology.

2 QUANTITATIVELY CHARACTERIZING THE RISK OF AIRBORNE TRANSMISSION IN SHARED-ROOM AIR

While Figure 1B is conceptually simple, to quantitatively capture the factors that control the probability of infection of a single susceptible individual *conditional* to the presence of one infector (which is the same as the attack rate) a more complex formulation is needed. Peng et al.[7] showed that the conditional probability of infection in shared room air can be rigorously calculated (within the approximations of the model) as a function of a single parameter, the relative risk parameter (H_r):

(1)

where r_E (r_B) is the enhancement of the shedding rate of infectious pathogen doses (breathing rate) for an activity with a certain degree of vocalization and physical intensity compared to sedentary breathing; f_e (f_i) is the penetration efficiency of pathogen-carrying particles through masks or face coverings for exhalation (inhalation); V is the volume of the space; λ is the first-order rate of removal of quanta (sum of λ_0 by ventilation with outdoor air; λ_{cle} air cleaning devices such as filters and ultraviolet disinfection; λ_{dec} decay of pathogen infectivity; and λ_{dep} aerosol deposition); D is the duration of exposure; and r_{ss} is an algebraic function of the above parameters that accounts for the approach to steady state conditions.

These authors also showed that the total number of new infections (i.e., the reproductive number of the event) is similarly a function of the risk parameter H:

(2)

where N_{sus} is the number of susceptible people present in the event.

In terms of characterizing a new potentially airborne disease, the attack rate of different superspreading events can be plotted versus H_r similar to Figure 1A. This provides a rapid empirical characterization of the potential of a given disease for shared-room airborne transmission. For example, for the wild type SARS-CoV-2, no outbreaks are observed for

$H_r \lesssim 0.001$ h^2 m^{-3}, the attack rate is below 20% for $H_r < 0.02$ h^2 m^{-3}, and larger outbreaks are observed only for $H_r > 0.05$ h^2 m^{-3}. These thresholds will vary for different diseases depending on their contagiousness.[7]

3 MAKING SPACES SAFER FROM SHARED-ROOM AIRBORNE TRANSMISSION

These results enable the possibility of rapidly estimating the risk of infection in shared-room air of different spaces in existing buildings, as well as characterizing it during planned construction of new buildings and refurbishment of existing buildings. Such assessments can inform mitigation and prevention of airborne or respiratory disease, including the planning of "airborne pandemic" or "high respiratory disease prevalence" modes of operation for different buildings.

Three critical parameters are needed for this purpose:

a. A characterization of each space and the human activities in it (by the values of H_r and H). An online calculator is available at **http://tinyurl.com/covid-estimator**. Note that H_r quantifies the relative risk assuming the presence of a single infector, while H incorporates the effect of the number of occupants on risk.
b. A list of mitigations that could be implemented in response to an outbreak of airborne disease (e.g., increasing outdoor air in the ventilation system, opening windows, distributing masks or respirators, installing portable filtration devices, reducing event duration, reducing occupancy, moving more intense vocalization and exercise outdoors), from which the reduced H_r (H) under each of those mitigations can be calculated.
c. A characterization of the disease, by a plot such as Figure **1A**.

The only parameters that may require some experimentation are quantifying the ventilation and filtration rates of each space, which can be accomplished in many cases with inexpensive portable CO_2 and particle meters.[23] With this information, the riskiest spaces can be quickly identified, as well as how much the risk is reduced by applying each of (and several or all of) the possible mitigation measures. This would allow quickly determining which spaces or activities should be temporarily suspended during periods of significant community transmission of a given airborne disease, vs. which ones can proceed with different degrees of mitigation, vs. which ones are low risk and do not require modifications.

For diseases that are known, for example, the current variant(s) of COVID-19, the classification could be carried out immediately, and the characterization of each space (by its H_r and H without and with different mitigations) would be known. However, detailed analysis of more shared-room airborne outbreaks for different diseases would be useful, as a surprisingly low

number have been documented in the literature. This is presumably due to the lack of expertise on ventilation from most public health professionals performing the large majority of investigations. Collaboration of building and/or aerosol scientists in future epidemiological investigations of potentially airborne superspreading events is critical.

For a new disease, for example, a COVID-19 variant that spreads differently from past ones, or the early propagation of a new pathogen with pandemic potential and suspected airborne transmission (most likely an influenza virus or coronavirus), preparation and rapid work could allow characterizing the potential of the pathogen for superspreading in shared-room air. As enough superspreading shared-room outbreaks were reported, public health teams investigating them could quickly report their secondary attack rate and the values of all the key parameters to a central point of contact (e.g., WHO), so that a plot such as Figure 1A could be constructed. This would have started to be possible in February to March 2020 for COVID-19, when multiple shared-room superspreading events had already been identified.[7] With that information the minimum value of H_r that appeared to allow significant superspreading could be identified and rapidly communicated. Then, the use and mitigation practices in different building spaces could be adapted for locations in which the disease was present, and depending on the incidence at that location. Together with other approaches such as publicly visible CO_2 monitors in riskier locations where air is shared (e.g., bars, restaurants, gyms, and choirs),[24] it would allow more rapid and efficient mitigation of the spread of new airborne diseases with pandemic potential.

ACKNOWLEDGEMENTS

We thank Zeynep Tufecki, Sandy Johnson, Dongwook Kim, and Harald Stark for useful discussions, and two anonymous reviewers for useful suggestions.

CONFLICT OF INTEREST

There is no conflicts of interest to disclose.

AUTHOR CONTRIBUTIONS

Jose L. Jimenez conceived the article and wrote the first draft with input from Zhe Peng. Demetrios Pagonis performed the measurements described in the Supplementary Information. All authors participated in revising the manuscript.

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The peer review history for this article is available at https://publons.com/publon/10.1111/ina.13025 .

Supporting Information

Filename	Description
ina13025-sup-0001-Supinfo.docx Word 2007 document , 1.2 MB	Supplementary Material

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